NCBI Entrez Nucleotide

PubMed Nucleotide Protein Genome Structure PMC Taxonomy OMIM Book

Search Nucleotide for human nr cam Go Clear

Limits Preview/Index History Clipboard Details

Display Summary Show: 20 Send to Text

Items 1-4 of 4 One page.

1: NM_002837 Links
Homo sapiens protein tyrosine phosphatase, receptor type, B (PTPRB), mRNA
gi|18491009|ref|NM_002837.2|[18491009]

2: BI793309 Links
ie50f01.y1 Melton Normalized Human Islet 4 N4-HIS 1 Homo sapiens cDNA clone IMAGE:5670289 5' similar to TR:Q9QVN3 Q9QVN3 R NR-CAM 12=NEUROFASCIN HOMOLOG/IG-FNIII CELL ADHESION MOLECULE {ALTERNATIVELY SPLICED}. ;, MRNA sequence
gi|15821034|gb|BI793309.1|[15821034]

3: N86739 Links
L0214F Human fetal heart, Lambda ZAP Express Homo sapiens cDNA clone L0214 5' similar to NEURONAL TRANSMEMBRANE PROTEIN NR-CAM, MRNA sequence
gi|1439941|gb|N86739.1|[1439941]

4: R25521 Links
yg44f06.r1 Soares infant brain 1NIB Homo sapiens cDNA clone IMAGE:35271 5' similar to SP:A43425 A43425 NR-CAM, BRAVO=CELL ADHESION MOLECULE L1 HOMOLOG ;, MRNA sequence
gi|781656|gb|R25521.1|[781656]

Revised: July 5, 2002.

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Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Term	Documents
HNR-CAM	0
HNR-CAMS	0
(HNR-CAM AND 2).USPT.	0
(L2 AND HNR-CAM).USPT.	0

Database: US Patents Full-Text Database
US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L4

Refine Search

Recall Text | Clear

Search History

DATE: Thursday, April 03, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L4	L2 and hnr-cam	0	L4
L3	L2 and nr-cam	0	L3
L2	@PRAY <= 1998 AND (human and cam)	1685	L2
L1	@PRAY <= 1998 AND (human and (nr cam or hnr cam))	0	L1

END OF SEARCH HISTORY

WEST

End of Result Set

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L5: Entry 14 of 14 File: USPT Nov 19, 1996

DOCUMENT-IDENTIFIER: US 5576423 A
TITLE: Antibodies to the slam protein expressed on activated T cells

Other Reference Publication (14):
Vincent P. Mauro, et al., "Homophilic and Heterophilic Binding Activities of Nr-CAM, a Nervous System Cell Adhesion Molecule," J. Cell Biol. 119:191-202, Oct. 1992.

WEST

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L5: Entry 13 of 14 File: USPT Jun 16, 1998

DOCUMENT-IDENTIFIER: US 5766922 A
TITLE: Functional ligands for the axonal cell rcognition molecule contactin

Detailed Description Text (4):
During development of the nervous systems, neurons are guided by secreted and cell bound molecules that provide both negative and positive cues. The experiments described in the examples of Sections 6.1 and 6.2 show that RPTP.beta., a receptor type protein tyrosine phosphatase, may provide such a signal by interacting with the axonal recognition molecule contactin. RPTP.beta. is a developmentally regulated protein that exists in three forms, one secreted and two membrane bound. The extracellular region of RPTP.beta. has a multidomain structure consisting of a CAH-like domain, a single FNIII repeat, and a long cysteine free spacer region. The complex structural nature of its extracellular region may result in a multifunctional protein that is able to interact with different proteins. As documented by the data shown herein, the CAH and the FNIII domains bind to at least two potential ligands present on neurons or glial cells. Functional expression cloning in COS7 cells and affinity purification with a specific affinity matrix followed by microsequencing enabled unequivocal identification of the cell recognition molecule contactin (F3/F11) as a neuronal ligand of RPTP.beta.. The interaction between contactin and RPTP.beta. is mediated via the CAH domain of the phosphatase, while the FNIII domain appears to bind to another molecule expressed on the surface of glial cells. It was previously shown that the secreted proteoglycan form of RPTP.beta. interacts with tenascin, N-CAM and Ng-CAM (Grumet et al., 1994, J. Biol. Chem., 269:12142-12146; Barnea et al., 1994, J. Biol. Chem., 269:14349-14352; Grumet et al., 1993, J. Cell. Biol., 120:815-724; Milev et al., 1994, J. Cell. Biol., 127:1703-1715). Since N-CAM and Ng-CAM do not bind directly to the CAH or the FNIII domain of RPTP.beta., they may interact with the large spacer domain of the phosphatase. Alternatively, they could interact with RPTP.beta. through a third component. Contactin may fulfill this function since it has been shown to interact with Ng-CAM, Nr-CAM, and the matrix proteins tenascin and restriction (Brummendorf et al., 1993, Neuron, 10:711-727; Morales et al., 1993, Neuron, 11:1113-1122; Zisch et al., 1992, J. Cell. Biol., 119:203-213). The various subdomains of the extracellular region of RPTP.beta. are able to interact with several distinct proteins that are expressed on diverse cell types in the central nervous system.

Detailed Description Text (115):
Contactin has been shown to be involved in both positive and negative responses of neurons to various stimuli (Brummendorf and Rathjen, 1993, J. Neurochem., 61:127-1219). When presented as a ligand to neurons, either as a membrane-bound or a soluble form, contactin induces axonal growth (Brummendorf et al., 1993, Neuron, 10:711-727; Clarke et al., 1993, Eur. J. Cell. Biol., 61:108-115; Durbec et al., 1992, J. Cell. Biol., 117:877-887; Gennarini et al., 1989, J. Cell. Biol., 109:755-788). Its neural receptor has been identified as the recognition molecule Nr-CAM (Morales et al., 1993, Neuron 11:1113-1122). On the other hand, contactin itself is a receptor present on neurons and mediates their repulsion by the extracellular matrix protein janusin (Pesheva et al., 1993, Neuron, 10:69-82). The results described in the example of Section 6.1 indicate that the CAH domain of RPTP.beta. can bind to contactin on cells. To analyze effects of this binding on neurons, chick tectal cells, known to express contactin, were plated on dishes previously coated with .beta.CF-Fc fusion protein or with Ng-CAM or laminin as controls. Cells attached and grow processes on both of these substrates (FIG. 7A). Treatment of the cells with PI-PLC prior to plating completely abolished cell attachment and neurite extension on RPTP.beta.. In

contrast, PI-PLC did not have a dramatic effect on cells growing on Ng-CAM or laminin as substrate (FIG. 7A). Thus, it was concluded that the CAH domain of RPTP.beta.is a permissive substrate for neuronal adhesion and neurite growth. Moreover, the cell adhesion and axonal elongation induced by RPTP.beta. is mediated through a GPI-anchored receptor.

WEST

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Search Results - Record(s) 1 through 10 of 14 returned.

1. Document ID: US 6541615 B1

L5: Entry 1 of 14 File: USPT Apr 1, 2003

US-PAT-NO: 6541615
DOCUMENT-IDENTIFIER: US 6541615 B1

TITLE: SIRP proteins and uses thereof

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

2. Document ID: US 6482605 B1

L5: Entry 2 of 14 File: USPT Nov 19, 2002

US-PAT-NO: 6482605
DOCUMENT-IDENTIFIER: US 6482605 B1

TITLE: Protein tyrosine phosphatase PTP20 and related products and methods

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

3. Document ID: US 6465210 B1

L5: Entry 3 of 14 File: USPT Oct 15, 2002

US-PAT-NO: 6465210
DOCUMENT-IDENTIFIER: US 6465210 B1

TITLE: Nucleic acid molecules encoding CASPR/p190

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

4. Document ID: US 6455026 B1

L5: Entry 4 of 14 File: USPT Sep 24, 2002

US-PAT-NO: 6455026
DOCUMENT-IDENTIFIER: US 6455026 B1

TITLE: Use of protein tyrosine phosphatase zeta as a biomolecular target in the

treatment and visualization of brain tumors

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 6399065 B1

L5: Entry 5 of 14 File: USPT Jun 4, 2002

US-PAT-NO: 6399065
DOCUMENT-IDENTIFIER: US 6399065 B1

TITLE: Methods for modulating SLAM-expressing T cells

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 6. Document ID: US 6372899 B1

L5: Entry 6 of 14 File: USPT Apr 16, 2002

US-PAT-NO: 6372899
DOCUMENT-IDENTIFIER: US 6372899 B1

TITLE: Purified genes encoding mammalian cell surface antigens; proteins and antibodies

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 7. Document ID: US 6313265 B1

L5: Entry 7 of 14 File: USPT Nov 6, 2001

US-PAT-NO: 6313265
DOCUMENT-IDENTIFIER: US 6313265 B1

TITLE: Neurite outgrowth-promoting polypeptides containing fibronectin type III repeats and methods of use

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

---

☐ 8. Document ID: US 6121231 A

L5: Entry 8 of 14 File: USPT Sep 19, 2000

US-PAT-NO: 6121231
DOCUMENT-IDENTIFIER: US 6121231 A

TITLE: Use of the KAL protein and treatment with the KAL protein in treatment of retinal, renal, neuromal and neural injury

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc
Image

☐ 9. Document ID: US 5977303 A

L5: Entry 9 of 14 File: USPT Nov 2, 1999

US-PAT-NO: 5977303
DOCUMENT-IDENTIFIER: US 5977303 A

TITLE: Mammalian cell surface antigens

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc
Image

☐ 10. Document ID: US 5846800 A

L5: Entry 10 of 14 File: USPT Dec 8, 1998

US-PAT-NO: 5846800
DOCUMENT-IDENTIFIER: US 5846800 A

TITLE: Nucleic acid molecules encoding a novel receptor-type protein tyrosine phosphatase-.sigma.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc
Image

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Term	Documents
NR-CAM.USPT.	14
NR-CAMS	0
NR-CAM.USPT.	14
(NR-CAM).USPT.	14

Display Format: TI | Change Format

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Search Results - Record(s) 11 through 14 of 14 returned.

☐ 11. Document ID: US 5840842 A

L5: Entry 11 of 14 File: USPT Nov 24, 1998

US-PAT-NO: 5840842
DOCUMENT-IDENTIFIER: US 5840842 A

TITLE: Receptor-type phosphotyrosine phosphatase-sigma

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 12. Document ID: US 5792743 A

L5: Entry 12 of 14 File: USPT Aug 11, 1998

US-PAT-NO: 5792743
DOCUMENT-IDENTIFIER: US 5792743 A

TITLE: Method for promoting neural growth comprising administering a soluble neural cell adhesion molecule

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 13. Document ID: US 5766922 A

L5: Entry 13 of 14 File: USPT Jun 16, 1998

US-PAT-NO: 5766922
DOCUMENT-IDENTIFIER: US 5766922 A

TITLE: Functional ligands for the axonal cell rcognition molecule contactin

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 14. Document ID: US 5576423 A

L5: Entry 14 of 14 File: USPT Nov 19, 1996

US-PAT-NO: 5576423
DOCUMENT-IDENTIFIER: US 5576423 A

TITLE: Antibodies to the slam protein expressed on activated T cells

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

Generate Collection | Print

Term	Documents
NR-CAM.USPT.	14
NR-CAMS	0
NR-CAM.USPT.	14
(NR-CAM).USPT.	14

Display Format: TI | Change Format

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              10  NR-CAM
          184694  CDNA
      S1       1  HUMAN AND NR-CAM AND CDNA
? t s1/3,ab/all

 1/3,AB/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

11492348   BIOSIS NO.: 199800273680
Cell adhesion molecule Nr-CAM is over-expressed in human brain
  tumors.
AUTHOR: Sehgal Anil(a); Boynton Alton L; Young Ronald F; Vermeulen Sandra S
  ; Yonemura Kenneth S; Kohler Erik P; Aldape Hector C; Simrell Charles R;
  Murphy Gerald P
AUTHOR ADDRESS: (a)Pacific Northwest Cancer Foundation, 120 Northgate
  Plaza, Room 230, Seattle, WA 98125**USA
JOURNAL: International Journal of Cancer  76 (4):p451-458 May 18, 1998
ISSN: 0020-7136
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Using the technique of differential display-polymerase chain
  reaction (DD-PCR), we isolated a cDNA fragment that is
  over-expressed in glioblastoma multiforme tissue as compared to normal
  brain tissue. Sequence analysis indicated that this sequence is identical
  to the previously isolated human neuron-glia-related cell adhesion
  molecule hNr-CAM. Gene-specific RT-PCR analysis indicated that hNr-CAM is
  over-expressed in high-grade astrocytomas, gliomas and glioblastoma tumor
  tissues as compared to  normal brain tissue. High levels of hNr-CAM
  expression also were observed in cell lines derived from astrocytomas,
  gliomas and glioblastoma multiforme tumors. Low levels of hNr-CAM
  expression were observed in neuroblastoma, meningiomas, melanoma, normal
  breast and prostate tumor tissues. Northern blot analysis showed an
  alternatively spliced mRNA of 1.4 kb in several tumors as compared to the
  7.5 kb transcript found in normal brain tissue. Genomic Southern blot
  analysis of DNA from 3 brain  tumor cell lines showed that
  over-expression of hNr-CAM in brain tumors was not due to gene
  amplification. In situ hybridization analysis indicated that 11 of the 20
  human brain tumor samples studied showed hNr-CAM over-expression.
  Our results suggest that hNr-CAM is over-expressed in malignant brain
  tumors and can serve as a novel marker for brain tumor detection and
  perhaps therapy.

1998
?
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not enabled for all types of tumors

3/21/05

12/14/98

4/27/99

1998
? s hnr-cam and (gene or cdna) and py<1999
Processing
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          1385426 GENE
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         21938101 PY<1999
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            16839 CAM
      S4   7 HNR(W)CAM
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...completed examining records
      S5   4 RD (unique items)
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 5/3,AB/1   (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

11369115   98249998   PMID: 9590116
  Cell adhesion molecule Nr-CAM is over-expressed in human brain tumors.
  Sehgal A; Boynton A L; Young R F; Vermeulen S S; Yonemura K S; Kohler E P
; Aldape H C; Simrell C R; Murphy G P
  Deke Slayton Center for Brain Cancer Studies, Pacific Northwest Cancer
Foundation, Northwest Hospital, Seattle, WA 98125, USA. asehgal@nwhsea.org
  International journal of cancer. Journal international du cancer (UNITED
STATES) May 18 1998, 76 (4) p451-8, ISSN 0020-7136 Journal Code:
0042124
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  Using the technique of differential display-polymerase chain reaction
(DD-PCR), we isolated a cDNA fragment that is over-expressed in
glioblastoma multiforme tissue as compared to normal brain tissue. Sequence
analysis indicated that this sequence is identical to the previously
isolated human neuron-glia-related cell adhesion molecule **hNr-
CAM**. Gene-specific RT-PCR analysis indicated that **hNr-CAM**
is over-expressed in high-grade astrocytomas, gliomas and glioblastoma
tumor tissues as compared to normal brain tissue. High levels of **hNr-
CAM** expression also were observed in cell lines derived from
astrocytomas, gliomas and glioblastoma multiforme tumors. Low levels of
hNr-CAM expression were observed in neuroblastoma, meningiomas,
melanoma, normal breast and prostate tumor tissues. Northern blot analysis
showed an alternatively spliced mRNA of 1.4 kb in several tumors as
compared to the 7.5 kb transcript found in normal brain tissue. Genomic
Southern blot analysis of DNA from 3 brain tumor cell lines showed that
over-expression of **hNr-CAM** in brain tumors was not due to gene
amplification. In situ hybridization analysis indicated that 11 of the 20
human brain tumor samples studied showed **hNr-CAM**
over-expression. Our results suggest that **hNr-CAM** is
over-expressed in malignant brain tumors and can serve as a novel marker
for brain tumor detection and perhaps therapy.

 5/3,AB/2   (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

11194960 98071552 PMID: 9407626
Molecular changes during the genesis of human gliomas.
Sehgal A
Deke Slayton Center for Brain Cancer Studies, Northwest Hospital, Seattle, Washington, USA. asehgal@nwhsea.org
Seminars in surgical oncology (UNITED STATES) Jan-Feb 1998, 14 (1) p3-12, ISSN 8756-0437 Journal Code: 8503713
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Neoplastic transformation in the normal human brain occurs as a result of the accumulation of a series of genetic alterations. These genetic alterations include the loss, gain or amplification of different chromosomes which lead to altered expression of proteins that play important roles in the regulation of cell proliferation. Several common genetic alterations at the chromosomal level (loss of 17p, 13q, 9p, 19, 10, 22q, 18q and amplification of 7 and 12q) have been observed. These alterations lead to changes in the expression of several genes; protein 53 (p53), retinoblastoma (RB), interferon (INF) alpha/beta, cyclic AMP dependent kinase number 2 (CDKN2), mutated in multiple advanced cancers 1 (MMAC1), deleted-in-colon carcinoma (DCC), epidermal growth factor receptor (EGFR), platelet derived growth factor (PDGF), platelet derived growth factor receptor (PDGFR), MDM2, GL1, CDK4 and SAS during the genesis and progression of human gliomas. Recent studies suggest that altered expression of several other genes [MET; MYC; transforming growth factor beta (TGF beta); CD44; vascular endothelial growth factor (VEGF); human neurological-related cell adhesion molecule (**hNr-CAM**); neuroglial cell adhesion molecule (NCAM L1); p21waf1/Cip1; TRKA; mismatch repair genes (MMR); C4-2; D2-2] and proteins [e.g., cathepsins, tenascin, matrix metalloproteases, tissue inhibitors of metalloproteases, nitric oxide synthase, integrins, interleukin-13 receptor (IL-13R), Connexin43, urokinase-type plasminogen activator receptors (uPARs), extracellular matrix proteins and heat shock proteins] are associated with the genesis of human gliomas. Taken together, these findings point to the accumulation of multiple genetic mutations coupled with extensive changes in gene expression in the etiology of human gliomas.

5/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08876702 20162602 PMID: 10697494
Antisense human neuroglia related cell adhesion molecule **hNr-CAM**, reduces the tumorigenic properties of human glioblastoma cells.
Sehgal A; Ricks S; Warrick J; Boynton A L; Murphy G P
Department of Neurological Surgery, University of California at San Francisco 94103, USA. sehgala@neurosurg.ucsf.edu
Anticancer research (GREECE) Nov-Dec 1999, 19 (6B) p4947-53, ISSN 0250-7005 Journal Code: 8102988
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: Human Nr-CAM (Neuroglia related Cell Adhesion Molecule) is over expressed in glioblastoma multiforme tissue (GMT) as compared to normal brain tissue (NBT). MATERIALS AND METHODS: We transfected a human glioblastoma cell line (2020-CRL) with a vector that overexpresses antisense **hNr-CAM** using a CMVpromoter. RESULTS: Antisense **hNr- CAM** caused reduction in the native **hNr-CAM** expression, changed cell morphology, reduced the cell proliferation rate and lengthening of the cell cycle. Furthermore, antisense **hNr-CAM** overexpression in these cells caused extensive reduction in the

number of soft agar colonies and invasion through extra cellular matrix (ECM) gel in vitro. Subcutaneous injection of antisense **hNr-CAM** overexpressing glioblastoma cells into nude mice caused complete inhibition of tumor formation as compared to vector only transfected cells. Intra-tumoral inoculation of antisense **hNr-CAM** expressing plasmid also caused slow tumor growth in nude mice in vivo. CONCLUSION: On the basis of these results, we conclude that **hNr-CAM** is a valid target for potential gene therapy of glioblastoma tumors.

5/3,AB/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11929133 BIOSIS NO.: 199900175242
Antisense **hNr-CAM** in the gene therapy of human glioblastoma tumors.
AUTHOR: Sehgal A; Boynton A; Ricks S; Warrick J; Murphy G P
AUTHOR ADDRESS: Pacific Northwest Cancer Found., Northwest Hosp., Seattle, WA 98125**USA
JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 40p437 March, 1999
CONFERENCE/MEETING: 90th Annual Meeting of the American Association for Cancer Research Philadelphia, Pennsylvania, USA April 10-14, 1999
SPONSOR: American Association for Cancer Research
ISSN: 0197-016X
RECORD TYPE: Citation
LANGUAGE: English
1999

AUTHORS Ratcliffe,C.F., Qu,Y., McCormick,K.A., Tibbs,V.C., Dixon,J.E., Scheuer,T. and Catterall,W.A.
TITLE A sodium channel signaling complex: modulation by associated receptor protein tyrosine phosphatase beta
JOURNAL Nat. Neurosci. 3 (5), 437-444 (2000)
MEDLINE 20233995
PUBMED 10769382
REFERENCE 7 (bases 1 to 6331)
AUTHORS Adamsky,K., Schilling,J., Garwood,J., Faissner,A. and Peles,E.
TITLE Glial tumor cell adhesion is mediated by binding of the FNIII domain of receptor protein tyrosine phosphatase beta (RPTPbeta) to tenascin C
JOURNAL Oncogene 20 (5), 609-618 (2001)
MEDLINE 21214455
PUBMED 11313993
COMMENT REVIEWED REFSEQ: This record has been curated by NCBI staff. The reference sequence was derived from X54131.1 and BE042873.1.
On Feb 4, 2002 this sequence version replaced gi:4506304.
Summary: The protein encoded by this gene is a member of the protein tyrosine phosphatase (PTP) family. PTPs are known to be signaling molecules that regulate a variety of cellular processes including cell growth, differentiation, mitotic cycle, and oncogenic transformation. This PTP contains an extracellular domain, a single transmembrane segment and one intracytoplasmic catalytic domain, thus belongs to receptor type PTP. The extracellular region of this PTP is composed of multiple fibronectin type_III repeats, which was shown to interact with neuronal receptor and cell adhesion molecules, such as contactin and tenascin C. This protein was also found to interact with sodium channels, and thus may regulate sodium channels by altering tyrosine phosphorylation status. The functions of the interaction partners of this protein implicate the roles of this PTP in cell adhesion, neurite growth, and neuronal differentiation.
COMPLETENESS: complete on the 3' end.

FEATURES Location/Qualifiers

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Revised: July 5, 2002.

1: NM_002837. Homo sapiens prot...[gi:18491009] Links

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VERSION     NM_002837.2  GI:18491009
KEYWORDS    .
SOURCE      Homo sapiens (human)
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REFERENCE   1  (bases 1 to 6331)
  AUTHORS   Kaplan,R., Morse,B., Huebner,K., Croce,C., Howk,R., Ravera,M.,
            Ricca,G., Jaye,M. and Schlessinger,J.
  TITLE     Cloning of three human tyrosine phosphatases reveals a multigene
            family of receptor-linked protein-tyrosine-phosphatases expressed
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  JOURNAL   Proc. Natl. Acad. Sci. U.S.A. 87 (18), 7000-7004 (1990)
  MEDLINE   90384936
   PUBMED   2169617
REFERENCE   2  (bases 1 to 6331)
  AUTHORS   Krueger,N.X., Streuli,M. and Saito,H.
  TITLE     Structural diversity and evolution of human receptor-like protein
            tyrosine phosphatases
  JOURNAL   EMBO J. 9 (10), 3241-3252 (1990)
  MEDLINE   91006018
   PUBMED   2170109
REFERENCE   3  (bases 1 to 6331)
  AUTHORS   Harder,K.W., Anderson,L.L., Duncan,A.M. and Jirik,F.R.
  TITLE     The gene for receptor-like protein tyrosine phosphatase (PTPRB) is
            assigned to chromosome 12q15-->q21
  JOURNAL   Cytogenet. Cell Genet. 61 (4), 269-270 (1992)
  MEDLINE   93137605
   PUBMED   1486802
REFERENCE   4  (bases 1 to 6331)
  AUTHORS   Peles,E., Nativ,M., Campbell,P.L., Sakurai,T., Martinez,R., Lev,S.,
            Clary,D.O., Schilling,J., Barnea,G., Plowman,G.D. et al.
  TITLE     The carbonic anhydrase domain of receptor tyrosine phosphatase beta
            is a functional ligand for the axonal cell recognition molecule
            contactin
  JOURNAL   Cell 82 (2), 251-260 (1995)
  MEDLINE   95354206
   PUBMED   7628014
REFERENCE   5  (bases 1 to 6331)
  AUTHORS   Sakurai,T., Lustig,M., Nativ,M., Hemperly,J.J., Schlessinger,J.,
            Peles,E. and Grumet,M.
  TITLE     Induction of neurite outgrowth through contactin and Nr-CAM by
            extracellular regions of glial receptor tyrosine phosphatase beta
  JOURNAL   J. Cell Biol. 136 (4), 907-918 (1997)
  MEDLINE   97201469
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DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

08876702   20162602   PMID: 10697494
   Antisense human neuroglia related cell adhesion molecule hNr-CAM,
reduces the tumorigenic properties of human glioblastoma cells.
  Sehgal A; Ricks S; Warrick J; Boynton A L; Murphy G P
  Department of Neurological Surgery, University of California at San
Francisco 94103, USA. sehgala@neurosurg.ucsf.edu
  Anticancer research (GREECE)  Nov-Dec 1999, 19 (6B) p4947-53, ISSN
0250-7005  Journal Code: 8102988
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  BACKGROUND: Human Nr-CAM (Neuroglia related Cell Adhesion
Molecule) is over expressed in glioblastoma multiforme tissue (GMT) as
compared to normal brain tissue (NBT). MATERIALS AND METHODS: We
transfected a human glioblastoma cell line (2020-CRL) with a vector that
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overexpresses **antisense** hNr-CAM using a CMVpromoter. RESULTS: **Antisense** hNr- CAM caused reduction in the native hNr-CAM expression, changed cell morphology, reduced the cell proliferation rate and lengthening of the cell cycle. Furthermore, **antisense** hNr-CAM overexpression in these cells caused extensive reduction in the number of soft agar colonies and invasion through extra cellular matrix (ECM) gel in vitro. Subcutaneous injection of **antisense** hNr-CAM overexpressing glioblastoma cells into nude mice caused complete inhibition of tumor formation as compared to vector only transfected cells. Intra-tumoral inoculation of **antisense** hNr-CAM expressing plasmid also caused slow tumor growth in nude mice in vivo. CONCLUSION: On the basis of these results, we conclude that hNr-CAM is a valid target for potential gene therapy of glioblastoma tumors.

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Set     Items   Description
S1          0   (NR (P) CAM) AND (ANTISENS? OR RIBOZYM?)
S2         94   NR AND CAM
S3         78   NR (W) CAM
S4          2   S3 AND (ANTISENS? OR RIBOZYM?)
S5          1   RD (unique items)
S6         14   S3 AND INHIBIT?
S7          7   RD (unique items)
S8         30   NEUROGLIA AND RELATED AND CELL AND ADHESION AND MOLECULE
? s s8 not s6
              30  S8
              14  S6
      S9      27  S8 NOT S6
? rd
...completed examining records
     S10      27  RD (unique items)
? s s10 and (antisens? or ribozym?)
              27  S10
           37389  ANTISENS?
            6427  RIBOZYM?
     S11       1  S10 AND (ANTISENS? OR RIBOZYM?)
? t s11/3,kab/all
>>>"KAB" is not a valid format name in file(s): 5, 155
? t s11/3,ab/all

 11/3,AB/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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11929133   BIOSIS NO.: 199900175242
Antisense hNr-CAM in the gene therapy of human glioblastoma tumors.
AUTHOR: Sehgal A; Boynton A; Ricks S; Warrick J; Murphy G P
AUTHOR ADDRESS:  Pacific Northwest Cancer Found., Northwest Hosp., Seattle,
  WA 98125**USA
JOURNAL: Proceedings of the American Association for Cancer Research Annual
Meeting  40p437 March, 1999
CONFERENCE/MEETING: 90th Annual Meeting of the American Association for
Cancer Research  Philadelphia, Pennsylvania, USA  April 10-14, 1999
SPONSOR: American Association for Cancer Research
ISSN: 0197-016X
RECORD TYPE: Citation
LANGUAGE: English
1999
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therapy of glioblastoma tumors.
? ds

Set	Items	Description
S1	0	(NR (P) CAM) AND (ANTISENS? OR RIBOZYM?)
S2	94	NR AND CAM
S3	78	NR (W) CAM
S4	2	S3 AND (ANTISENS? OR RIBOZYM?)
S5	1	RD (unique items)

? s s3 and inhibit?

	78	S3
	2102101	INHIBIT?
S6	14	S3 AND INHIBIT?

? rd
...completed examining records

S7	7	RD (unique items)

? t s7/3,ab/all

7/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11822581 99262361 PMID: 10328925

Nr-CAM promotes neurite outgrowth from peripheral ganglia by a mechanism involving axonin-1 as a neuronal receptor.

Lustig M; Sakurai T; Grumet M
Department of Pharmacology, NYU Medical Center, 550 First Avenue, New York, New York, 10016, USA.

Developmental biology (UNITED STATES) May 15 1999, 209 (2) p340-51, ISSN 0012-1606 Journal Code: 0372762
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Nr-CAM is a neuronal cell adhesion molecule (CAM) belonging to the immunoglobulin superfamily that has been implicated as a ligand for another CAM, axonin-1, in guidance of commissural axons across the floor plate in the spinal cord. **Nr-CAM** also serves as a neuronal receptor for several other cell surface molecules, but its role as a ligand in neurite outgrowth is poorly understood. We studied this problem using a chimeric Fc-fusion protein of the extracellular region of **Nr-CAM** (Nr-Fc) and investigated potential neuronal receptors in the developing peripheral nervous system. A recombinant **Nr-CAM**-Fc fusion protein, containing all six Ig domains and the first two fibronectin type III repeats of the extracellular region of **Nr-CAM**, retains cellular and molecular binding activities of the native protein. Injection of Nr-Fc into the central canal of the developing chick spinal cord in ovo resulted in guidance errors for commissural axons in the vicinity of the floor plate. This effect is similar to that resulting from treatment with antibodies against axonin-1, confirming that axonin-1/**Nr-CAM** interactions are important for guidance of commissural axons through a spatially and temporally restricted **Nr-CAM** positive domain in the ventral spinal cord. When tested as a substrate, Nr-Fc induced robust neurite outgrowth from dorsal root ganglion and sympathetic ganglion neurons, but it was not effective for tectal and forebrain neurons. The peripheral but not the central neurons expressed high levels of axonin-1 both in vitro and in vivo. Moreover, antibodies against axonin-1 **inhibited** Nr-Fc-induced neurite outgrowth, indicating that axonin-1 is a neuronal receptor for **Nr-CAM** on these peripheral ganglion neurons. The results demonstrate a role for **Nr-CAM** as a ligand in axon growth by a mechanism involving axonin-1 as a neuronal receptor and suggest that dynamic changes in **Nr-CAM** expression can modulate axonal growth and guidance during development. Copyright 1999 Academic Press.

7/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10850152 97201469 PMID: 9049255
Induction of neurite outgrowth through contactin and **Nr-CAM** by extracellular regions of glial receptor tyrosine phosphatase beta.
Sakurai T; Lustig M; Nativ M; Hemperly J J; Schlessinger J; Peles E; Grumet M
Department of Pharmacology, New York University Medical Center 10016, USA.
Journal of cell biology (UNITED STATES) Feb 24 1997, 136 (4) p907-18, ISSN 0021-9525 Journal Code: 0375356
Contract/Grant No.: NS21629; NS; NINDS; NS33921; NS; NINDS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Receptor protein tyrosine phosphatase beta (RPTPbeta) is expressed as soluble and receptor forms with common extracellular regions consisting of a carbonic anhydrase domain (C), a fibronectin type III repeat (F), and a unique region called S. We showed previously that a recombinant Fc fusion protein with the C domain (beta C) binds to contactin and supports neuronal adhesion and neurite growth. As a substrate, betaCFS was less effective in supporting cell adhesion, but it was a more effective promoter of neurite outgrowth than betaCF. betaS had no effect by itself, but it potentiated neurite growth when mixed with betaCF. Neurite outgrowth induced by betaCFS was **inhibited** by antibodies against **Nr-CAM** and contactin, and these cell adhesion molecules formed a complex that bound betaCFS. NIH-3T3 cells transfected to express betaCFS on their surfaces induced neuronal differentiation in culture. These results suggest that binding of glial RPTPbeta to the contactin/**Nr-CAM** complex is important for neurite growth and neuronal differentiation.

7/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10166704 22170691 PMID: 12183361
Nr-CAM is a target gene of the beta-catenin/LEF-1 pathway in melanoma and colon cancer and its expression enhances motility and confers tumorigenesis.
Conacci-Sorrell Maralice E; Ben-Yedidia Tamar; Shtutman Michael; Feinstein Elena; Einat Paz; Ben-Ze'ev Avri
Department of Molecular Cell Biology, The Weizmann Institute of Science, Rehovot 76100, Israel.
Genes & development (United States) Aug 15 2002, 16 (16) p2058-72, ISSN 0890-9369 Journal Code: 8711660
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
beta-catenin and plakoglobin (gamma-catenin) are homologous molecules involved in cell adhesion, linking cadherin receptors to the cytoskeleton. beta-catenin is also a key component of the Wnt pathway by being a coactivator of LEF/TCF transcription factors. To identify novel target genes induced by beta-catenin and/or plakoglobin, DNA microarray analysis was carried out with RNA from cells overexpressing either protein. This analysis revealed that **Nr-CAM** is the gene most extensively induced by both catenins. Overexpression of either beta-catenin or plakoglobin induced **Nr-CAM** in a variety of cell types and the

LEF/TCF binding sites in the **Nr-CAM** promoter were required for its activation by catenins. Retroviral transduction of **Nr-CAM** into NIH3T3 cells stimulated cell growth, enhanced motility, induced transformation, and produced rapidly growing tumors in nude mice. **Nr-CAM** and LEF-1 expression was elevated in human colon cancer tissue and cell lines and in human malignant melanoma cell lines but not in melanocytes or normal colon tissue. Dominant negative LEF-1 decreased **Nr-CAM** expression and antibodies to **Nr-CAM** **inhibited** the motility of B16 melanoma cells. The results indicate that induction of **Nr-CAM** transcription by beta-catenin or plakoglobin plays a role in melanoma and colon cancer tumorigenesis, probably by promoting cell growth and motility.

7/3,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09779578 21585387 PMID: 11728309
Nr-CAM and neurofascin interactions regulate ankyrin G and sodium channel clustering at the node of Ranvier.
Lustig M; Zanazzi G; Sakurai T; Blanco C; Levinson S R; Lambert S; Grumet M; Salzer J L
Department of Pharmacology, New York University Medical Center, 550 First Avenue, New York, NY 10016, USA.
Current biology - CB (England) Nov 27 2001, 11 (23) p1864-9, ISSN 0960-9822 Journal Code: 9107782
Contract/Grant No.: 5T32 GM07308; GM; NIGMS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Voltage-dependent sodium (Na(+)) channels are highly concentrated at nodes of Ranvier in myelinated axons and play a key role in promoting rapid and efficient conduction of action potentials by saltatory conduction. The molecular mechanisms that direct their localization to the node are not well understood but are believed to involve contact-dependent signals from myelinating Schwann cells and interactions of Na(+) channels with the cytoskeletal protein, ankyrin G. Two cell adhesion molecules (CAMs) expressed at the axon surface, **Nr-CAM** and neurofascin, are also linked to ankyrin G and accumulate at early stages of node formation, suggesting that they mediate contact-dependent Schwann cell signals to initiate node development. To examine the potential role of **Nr-CAM** in this process, we treated myelinating cocultures of DRG (dorsal root ganglion) neurons and Schwann cells with an **Nr-CAM**-Fc (Nr-Fc) fusion protein. Nr-Fc had no effect on initial axon-Schwann cell interactions, including Schwann cell proliferation, or on the extent of myelination, but it strikingly and specifically **inhibited** Na(+) channel and ankyrin G accumulation at the node. Nr-Fc bound directly to neurons and clustered and coprecipitated neurofascin expressed on axons. These results provide the first evidence that neurofascin plays a major role in the formation of nodes, possibly via interactions with **Nr-CAM**.

7/3,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08876702 20162602 PMID: 10697494
Antisense human neuroglia related cell adhesion molecule hNr-CAM, reduces the tumorigenic properties of human glioblastoma cells.
Sehgal A; Ricks S; Warrick J; Boynton A L; Murphy G P
Department of Neurological Surgery, University of California at San

Francisco 94103, USA. sehgala@neurosurg.ucsf.edu
Anticancer research (GREECE) Nov-Dec 1999, 19 (6B) p4947-53, ISSN 0250-7005 Journal Code: 8102988
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: Human **Nr-CAM** (Neuroglia related Cell Adhesion Molecule) is over expressed in glioblastoma multiforme tissue (GMT) as compared to normal brain tissue (NBT). MATERIALS AND METHODS: We transfected a human glioblastoma cell line (2020-CRL) with a vector that overexpresses antisense hNr-CAM using a CMVpromoter. RESULTS: Antisense hNr- CAM caused reduction in the native hNr-CAM expression, changed cell morphology, reduced the cell proliferation rate and lengthening of the cell cycle. Furthermore, antisense hNr-CAM overexpression in these cells caused extensive reduction in the number of soft agar colonies and invasion through extra cellular matrix (ECM) gel in vitro. Subcutaneous injection of antisense hNr-CAM overexpressing glioblastoma cells into nude mice caused complete **inhibition** of tumor formation as compared to vector only transfected cells. Intra-tumoral inoculation of antisense hNr-CAM expressing plasmid also caused slow tumor growth in nude mice in vivo. CONCLUSION: On the basis of these results, we conclude that hNr-CAM is a valid target for potential gene therapy of glioblastoma tumors.

7/3,AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07538571 92407033 PMID: 1527169
Homophilic and heterophilic binding activities of **Nr-CAM**, a nervous system cell adhesion molecule.
Mauro V P; Krushel L A; Cunningham B A; Edelman G M
Rockefeller University, New York 10021.
Journal of cell biology (UNITED STATES) Oct 1992, 119 (1) p191-202, ISSN 0021-9525 Journal Code: 0375356
Contract/Grant No.: HD-09635; HD; NICHD; HD16550; HD; NICHD; NS-28932; NS ; NINDS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Nr-CAM is a membrane glycoprotein that is expressed on neurons. It is structurally related to members of the N-CAM superfamily of neural cell adhesion molecules having six immunoglobulin-like domains and five fibronectin type III repeats in the extracellular region. We have found that the aggregation of chick brain cells was **inhibited** by anti-**Nr-CAM** Fab' fragments, indicating that **Nr-CAM** can act as a cell adhesion molecule. To clarify the mode of action of **Nr-CAM** , a mouse fibroblast cell line L-M(TK-) (or L cells) was transfected with a DNA expression construct encoding an entire chicken **Nr-CAM** cDNA sequence. After transfection, L cells expressed **Nr-CAM** on their surface and aggregated. Aggregation was specifically **inhibited** by anti-**Nr-CAM** Fab' fragments. To check the specificity of this aggregation, a fusion protein (FGTNr) consisting of glutathione S-transferase linked to the six immunoglobulin domains and the first fibronectin type III repeat of **Nr-CAM** was expressed in Escherichia coli. Addition of FGTNr to the transfected cells blocked their aggregation. Further analysis using a combination of cell aggregation assays, binding of cells to FGTNr-coated substrates, aggregation of FGTNr-coated Covaspheres and binding of FGTNr-coated Covaspheres to FGTNr-coated substrates revealed that **Nr-CAM** mediates two types of cell interactions: a homophilic, divalent cation-independent binding, and a heterophilic, divalent cation-dependent

binding. Homophilic binding was demonstrated between transfected L cells, between chick embryo brain cells and FGTNr, and between Covaspheres to which FGTNr was covalently attached. Heterophilic binding was shown to occur between transfected and untransfected L cells, and between FGTNr and primary chick embryo fibroblasts; in all cases, it was dependent on the presence of either calcium or magnesium. Primary chick embryo glia or a human glial cell line did not bind to FGTNr-coated substrates. The results indicate that **Nr-CAM** is a cell adhesion molecule of the nervous system that can bind by two distinct mechanisms, a homophilic mechanism that can mediate interactions between neurons and a heterophilic mechanism that can mediate binding between neurons and other cells such as fibroblasts.

7/3,AB/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07445906 92309438 PMID: 1377280
Structure, expression, and function of Ng-CAM, a member of the immunoglobulin superfamily involved in neuron-neuron and neuron-glia adhesion.
Grumet M
Department of Developmental and Molecular Biology, Rockefeller University, New York, New York.
Journal of neuroscience research (UNITED STATES) Jan 1992, 31 (1) p1-13, ISSN 0360-4012 Journal Code: 7600111
Contract/Grant No.: HD-16550; HD; NICHD; NS-21629; NS; NINDS
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The neuron-glia cell adhesion molecule (Ng-CAM) mediates neuron-neuron adhesion by a homophilic mechanism and neuron-astrocyte adhesion by a heterophilic mechanism. The protein is expressed on neurons and Schwann cells but not on astrocytes. It is most prevalent during development on cell bodies of migrating neurons and on axons during formation of nerves. Ng-CAM expression is greatly increased following nerve injury. Anti-Ng-CAM antibodies **inhibited** migration of granule cells along Bergmann glia in cerebellar explants and fasciculation of neurites in outgrowths from explants of dorsal root ganglia. The combined results indicate that Ng-CAM on neurons binds to Ng-CAM on adjacent neurons and to as yet unidentified ligands on astrocytes. Ng-CAM is synthesized in chicken neurons from a 6 kb mRNA as Mr approximately 200,000 forms which are cleaved to yield two components of Mr 135,000 and 80,000. It is glycosylated and can be phosphorylated. Amino acid sequence analysis indicates that it contains six immunoglobulin domains, five fibronectin type III repeats, a transmembrane domain and a cytoplasmic region. Structural analyses indicate that Ng-CAM is most closely related to the mammalian glycoprotein L1 but significant differences between them strongly suggest that they are not equivalent molecules. The recent identification of another structurally related molecule in the chicken called **Nr-CAM** underscores the notion that these molecules are members of a subfamily of neural cell adhesion molecules within the immunoglobulin superfamily that have related or complementary functions in the nervous system.